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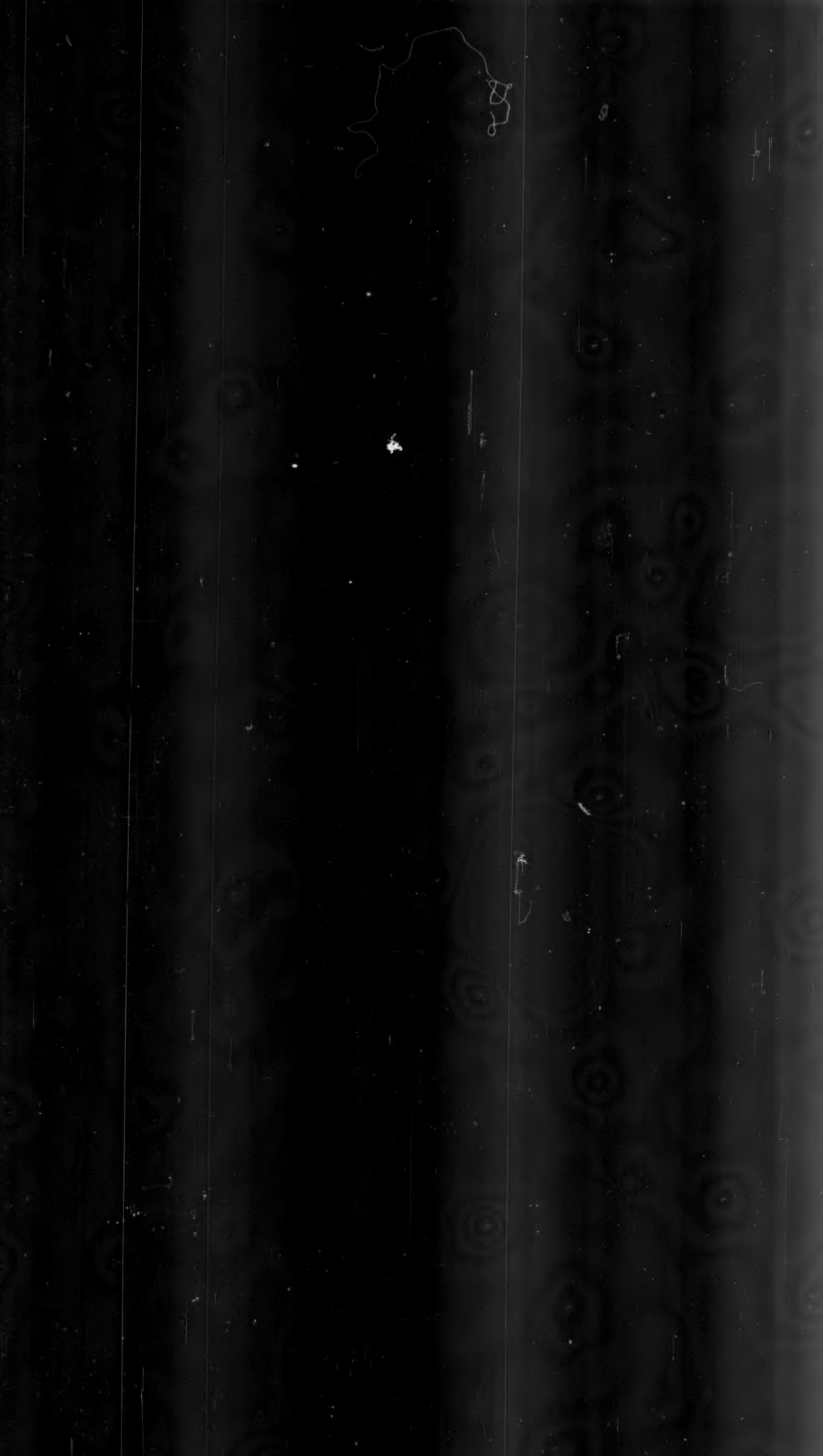


SCIATICA

AN ANATOMICAL AND MECHANICAL STUDY OF THE LUMBOSACRAL REGION

BY

NORMAN CAPENER

Princess Elizabeth Orthopaedic Hospital, Exeter

Great interest is being shown in sciatica at the present time. It is a common disturbance of obscure etiology. Its association with rheumatism and trauma renders it a severe economic problem, in war as in peace. An advance in the understanding of some forms of sciatica has been made in the recognition of the important part played by intervertebral disc lesions (Alajouanine and Petit-Dutaillis, 1930; Mixter and Barr, 1934; Peet and Echols, 1934). A certain amount of clinical research is being conducted upon special groups of cases. It is probable that other and more extensive investigations will be embarked upon as soon as circumstances permit. Therefore I have felt that the time is appropriate for the publication of certain researches into the anatomical and mechanical background of the problem.* Very little originality is claimed for the greater part of the work. Material (both clinical and radiological) has been studied and is here presented in the hope that it may prove a guide for investigators. Written as it is from the orthopaedic point of view, it is natural that the mechanical factors should be stressed rather than the pathological. I have tried to avoid glaring excursions into theoretic considerations, but it is impossible to avoid them altogether.

The Normal Lumbar Curve

The lumbar hollow is a human characteristic which babies acquire in the first year of life (Fig. 1). It is dependent upon extension of the thighs, which before birth have been held flexed upon the abdomen. After birth some " fixed " flexion of the hips remains. Extension force applied to these joints in small babies is communicated to the lumbar spine and a lordotic concavity is developed. Willis (1932) has suggested that the deleterious effects of such force upon the lumbar spine are aggravated in civilized people by " the efficient bathing, diapering, and swaddling attentions of the modern nurse." Certainly lordosis and protuberant abdomen are normal features in infants who are learning to walk; the relation of this in some children to lack of hip extension is to be remembered. Later, as true extension of the hip-joints occurs, there is shortening of the ham-string muscles, the influence of which, by tilting the pelvis backwards, is to reduce the lumbar lordosis. Any circumstance which unduly increases ham-string tension will restore the prenatal type of lumbar convexity backwards (Fig. 1, D). This is by no means an unusual circumstance, for it is to be found in any person who chooses to sit upright with his knees extended; or, with the knees extended, to pick up something from the ground. As we shall see later, the latter position is fraught with danger for those prone to sciatica, and the former is one all too frequently indulged in by those who spend too much time in bed. It is more than of passing interest that while the prenatal condition of the foetal lumbar spine is one of backward convexity, so, as Lambrinudi (1934) observed, is that of its mother's; which is not lordotic as so commonly thought. The condition of the gravid woman's

* Based on a Hunterian Lecture, " The Lumbo-sacral Joint: a Discussion upon Spondylolisthesis, Scoliosis, and Sciatica," Royal College of Surgeons; Jan. 22, 1941.

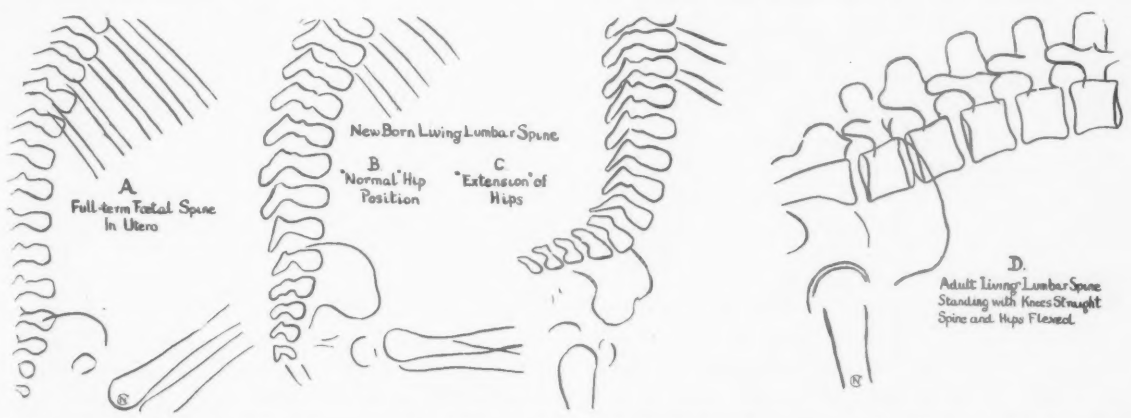


FIG. 1. The Lumbosacral Paradox. Vulnerability in infancy by deviation from prenatal curvature—i.e. by extension force. Vulnerability in adult life by return to prenatal type of curvature—i.e. by flexion force.

lumbar spine from the sixth month onwards is favourable for the development of sciatica.

Lumbar Spine as a Unit

The Question of Rotation.—Much of anatomical description in textbooks is directed to the varying features of individual segments, their shape, attachments and articulation. Too little attention, however, is paid to the spine as a whole, or to its regions as units of the whole. The spine is a concatenation and behaves like a chain. Movement of one segment involves movement of the whole. The periodicity of scoliosis, which so closely resembles a phase of vibration in a monochord, has its counterpart in the twists and curves of unstable pillars and towers. This is too big a subject to go into here, but if we do realize the obvious idea of the chain, it will be much easier to understand the question of whether rotation movements occur in the lumbar spine. Many writers still quote old textbook descriptions denying it, and J. E. Frazer still repeats that the articular facets of the lumbar vertebrae are so placed as to prevent rotation, which is only considered as being around a vertical axis situated in the centre of the vertebral bodies (Fig. 4). If, however, we disregard the segmental idea, there is no reason at all why the axis of rotation should not be found anywhere, even in mid-air. Certainly, from a mechanical viewpoint, one would expect it to lie on the concave side of the normal curve (as it does in the thoracic region). To everyone familiar with the problems of scoliosis it is obvious that a considerable degree of rotation does occur in the lumbar region. If the axis for this movement is regarded as being posterior to the spinal canal, then it is quite easy to see how the arrangement of the lumbar articular facts readily permits it. The range need be less than eight degrees between each segment to permit a total of forty-five degrees between the chest and the pelvis, which is probably as much as a normally supple person requires, and is more than he could produce in the thoracic region, which, relatively, has far less rotation because of the need for protection of its viscera. Owing to the more coronal arrangement of the lumbo-sacral facets it is probable that a larger degree of rotation is possible here than in the rest of the lumbar region. There can be no doubt about this matter of rotation for another reason—because it is a physical necessity in any lateral flexion. Bending sideways any structure like the lumbar spine, which has a pre-existing forward convexity, must be accompanied by torsion of the whole region (or rotation between individual segments).

The posterior neural arches and spinous processes are of greater mechanical importance than has generally been realized. Under the heading of " weight transmission " I shall have more to say, but here one should remark on an interesting phenomenon in the movement of hyper-extension: not only does weight tend to be transferred to the articular and spinous processes, but individual vertebrae tend to slide backwards on the one below. It is more readily detected in the upper lumbar region, and was well shown in the extreme example of lumbar hyperextension in a contorsionist whose radiograph was described by Lockhart and reproduced in the last edition of Cunningham's *Textbook of Anatomy*. I have demonstrated the same point in radiographs of normal spines (Fig. 2), and (Capener, 1932) in a remarkable case of spinal tuberculosis

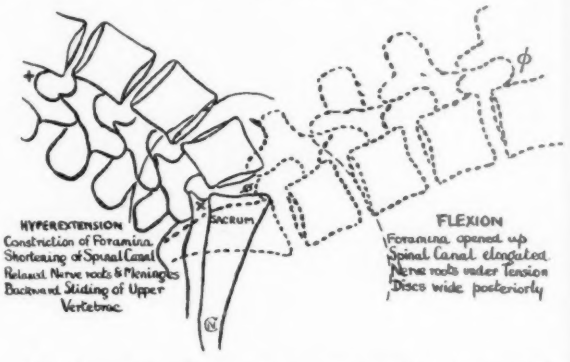


FIG. 2. Two views of the same spine in standing position. In the original films the distance between the same points marked X in extension and ø in flexion were $5\frac{5}{8}$ in. and $7\frac{5}{8}$ in. respectively.

associated with spondylolisthesis (Fig. 5). In the upper lumbar spine it may become a fixed condition and is then known as retrospondylolisthesis; being often due to post-traumatic degeneration of the intervertebral disc, or osteoarthritis.

Fourth Lumbar Vertebra

The importance of this vertebra in the present discussion is that it is the " spear head of attack " by superimposed body weight upon the mechanical weaknesses of the fifth lumbar vertebra. It is often the last vertebra of completely lumbar type. From the intervertebral disc beneath it there occurs posterior herniation with almost equal frequency as from the lumbo-sacral disc. In the past, prominence has been given to the various forms of true congenital anomaly of the fifth lumbar vertebra, such as complete or partial sacralization. Generally, attention has been paid to the aberrant masses which project laterally from one or other transverse process and articulate more or less completely with the lateral mass of the sacrum. Probably osteoarthritic changes can occur at these additional joints, but what tends to be overlooked is the consequent altered mechanical conditions in the joints between the fourth and fifth lumbar vertebrae. With the greater fixation of the fifth, the stresses to which the fourth is exposed without doubt explain such a circumstance as the greater liability then for the fourth to show spondylolytic changes, and of its intervertebral disc to suffer from trauma.

Intervertebral Discs

From a study of recent literature, it is obvious that these structures are of considerable significance in any discussion of sciatica. The best work upon the normal and morbid anatomy is that by Schmorl, made readily available to English readers by Beadle

(1931). I shall concentrate upon mechanical features.

Each disc is composed of two elements: (1) an annulus fibrosus, which is thinner posteriorly, and encloses (2) the nucleus pulposus, a somewhat spherical structure under tension, which is partly given to it by the encasing fibres of the annulus. The nucleus lies behind the centre of the disc, its position being recognized in a lateral radiograph by the slight hollow above and below in the outlines of the contiguous vertebral bodies. At the periphery each disc is bound down to the intervertebral ligaments, especially in front to the anterior common ligament, and to the slighter posterior common ligament behind. During movements of the spine it is apparent that change of shape must occur in each disc. In flexion and extension (Fig. 2) compression of one edge is accompanied by expansion of the opposite side. In lateral flexion and rotation (Fig. 3), and one cannot occur without the other,

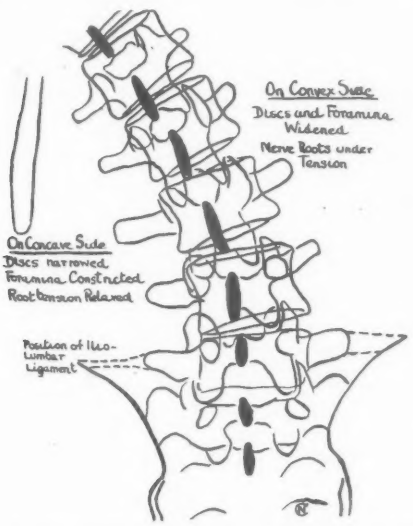


FIG. 3. Another standing spine showing lateral flexion and rotation.

considerable distortion must occur. In hyperextension a special sort of shearing effect must be present to allow of the backward sliding of the upper vertebrae already noted. A study of radiographs of the same spine, in different phases of movement, suggests that the nucleus pulposus is the pivot around which the more simple movements occur—in fact, rather like a see-saw. Such radiographs also can tell us something about the internal stresses of the vertebral bodies (Fig. 5). Where a portion of the disc is narrowed the parts of the vertebra above and below must also be in a state of compression, and when the disc is widened so the vertebral substance above and below must be in relative tension.

Let us consider posterior protrusion into the spinal canal of annulus or nuclear substance. One should realize that the annulus is weakest at this point, and that in flexion of the lumbar spine the internal stresses within the disc are relatively tensile or negative posteriorly, and predominantly compressive or positive anteriorly. The whole tendency, therefore, in forced lumbar flexion is for the nuclear substance to be expressed backwards like a "pea out of its pod." Any movement in the direction of flexion of the lumbar spine with the hips fixed in *relative* extension, as when the knees are straight, will, for reasons already mentioned, convey a greater flexion force to this part of the spine—a

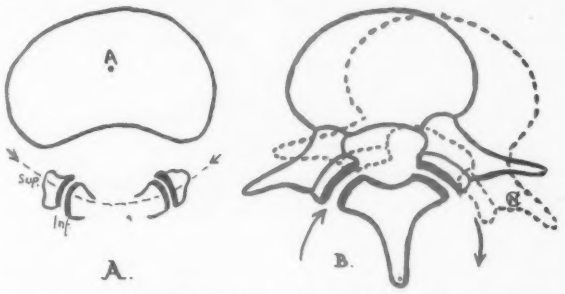


FIG. 4. Lumbar Rotation. A.—After Fraser who states there is none. B.—Author's interpretation (diagrammatic).

force which will be increased from above should the individual be lifting a weight. So important is the danger of this position regarded by certain American industries that placards are exhibited in workshops discouraging it and advising workmen to bend their knees and so "get under their weights." For a similar reason the habitual position of the invalid

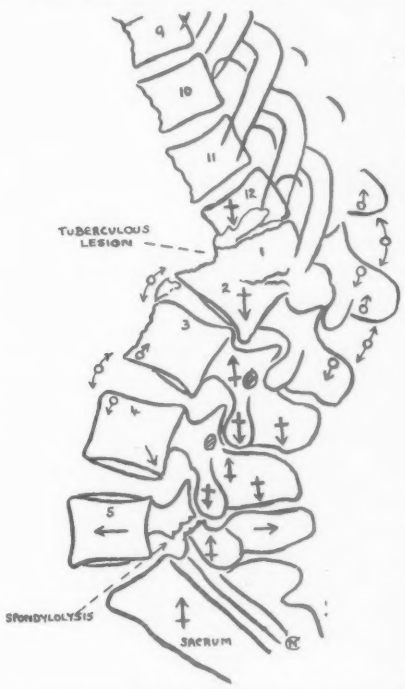


FIG. 5.—A demonstration of stresses in a pathological spine.

○ = tension. + = compression. Arrows indicate direction of force or movement.

who sits in bed with knees straight is also dangerous. An important point, noted by Schmorl, is the frequency with which disc protrusions within the spinal canal are found at autopsy; it seems that this is consistent with freedom from sciatic pain Whether a retropulsed nucleus can slip partially backwards

again is open to doubt. It certainly would seem unlikely that it could return completely. The idea of spontaneous reduction is attractive to the surgeon who at operation finds nothing when lipiodol radiography has been clearly positive. In such circumstances it should be recognized that the influence of weight-bearing is removed and that in recumbency there is a natural tendency for the intervertebral discs to resume a normal shape. It would be interesting to know if the amount of retropulsion is a variable phenomenon in any particular case.

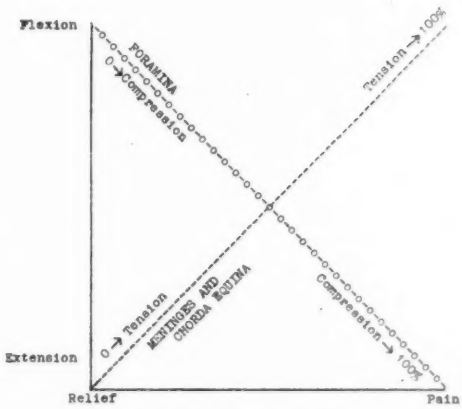
The Meninges and Intervertebral Foramina

In the spinal canal the meningeal tube is separated from its bony and ligamentous surroundings by an appreciable layer of extradural fatty areolar tissue which permits easy movement. The amount of this movement requires further attention, for it is surprising to most people.

If one compares lateral radiographs of the lumbar spine in full flexion with those of the same spine in hyperextension, the disparity in length is remarkable. In the illustrations (Fig. 2) showing this, measurements were taken between the two same points in the canal, and the elongation in full flexion is found to be at least one-third of the shorter length in hyperextension. Obviously, in flexion the meninges and cauda equina are under relative tension. It is the same position of danger for intervertebral discs and nuclei, so that the retropulsive tendencies of them are met by nerve tissues on the stretch. Conversely, in hyperextension relaxed meninges and nerves go with closed-up posterior disc spaces. While discussing the effect of lumbar movement and posture upon the intraspinal soft tissues one should mention changes occurring within the intervertebral foramina. First of all, there is interest in the feature to which attention was drawn by Danforth and Wilson (1925). Throughout the lumbar series these foramina become progressively smaller from above downwards to the lumbo-sacral interspace. This, which transmits the largest nerve root of them all, has the smallest available space. We must realize that each foramen also contains meningeal blood vessels and a prolongation of dura which eventually merges with the perineural sheath. Furthermore, each nerve root, which has emerged at an angle from beneath the pedicle above, has a variable degree of prolongation of arachnoid with cerebro-spinal fluid (Capener, 1937) (Fig. 6). This extension of the theca, particularly on the lower lumbar nerve roots, is generally not appreciated.

In full flexion of the lumbar spine the foramina are enlarged to their maximum, in hyperextension they are smallest (Fig. 2). The vulnerability of structures within them is greatest in hyperextension, which is the reverse to the dangers to structures within the spinal canal. In these altered relations we have, I believe, some explanation for the conflicting signs that many patients present. The diagram shows this graphically (Chart 1). In some patients relief from pain is given by hyperextension; in others, by flexion. In the former, relief may be due to relaxation of the cauda equina and meningeal tension or of nerve-root kink; while in flexion relief may result because there is greater room for the foraminal contents. On the contrary, flexion

	Flexion	Extension
Meninges and Chorda	Tension	Relaxed
Foramina	Wide open	Constricted
Intervertebral Disc Posterior Anterior	 Widened Narrowed	 Narrowed Widened



Simple Graph to explain why in certain types of Sciatica no satisfactory relief is given by change of position in the direction of flexion or extension. In these lateral flexion (Scoliosis) is necessary.

Chart 1

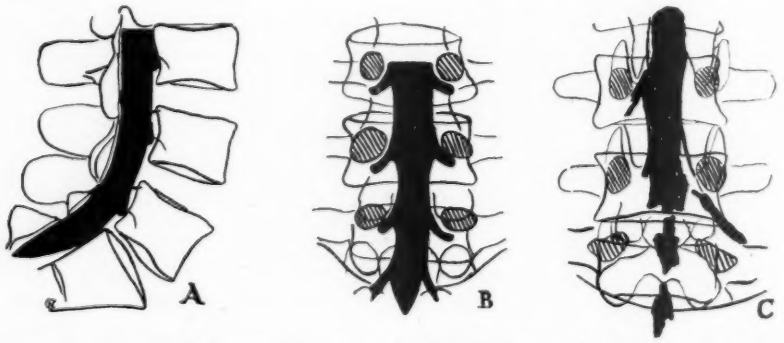


FIG. 6. Relation of nerve roots, pedicles and intervertebral foramina. A and B.—Autopsy specimen in which theca has been filled with lipiodol. C.—Radiograph taken in course of investigation of a case of nuclear retropulsion.

may increase disc and nuclear retropulsion, while by hyperextension one may aggravate pain by constricting foraminal contents. Lateral flexion will have a similar effect on the concave side, and rotation will increase nerve-root tension on one side. The constriction of intervertebral foraminal contents is, of course, of more significance should the nerve root be oedematous, or if the size of the foramen is further reduced by lateral disc protrusion, osteoarthritic outgrowth, and so forth. Here it should be noted that herniation of disc substance into a foramen is not seen in opaque myelography. Some of the most distressing forms of sciatica are in individuals who get relief by no alteration in position, and in whom every movement is agonizing. It will readily be appreciated from the above discussion how movement giving relief from one element of the painful condition is vitiated by the aggravation of something else. Nevertheless, posture does sometimes produce the most astonishing relief. When dealing with muscular function later on I shall have more to say on this; but here one should remark upon the fact that hyperextension occasionally is very helpful. It suggests that a nerve root, caught upon the summit of a bulging intervertebral disc, may be dislodged into the neighbouring valley by the backward sliding tendency of the upper vertebra, at a time when the chorda equina and meningeal tension is eased. Sudden jerks may do the same thing.

A phenomenon which illustrates the complexities of spinal mechanics is the peculiar scoliotic displacement of the trunk which is almost invariably associated with sciatica. Most frequently such displacement is to the opposite side (contralateral) and probably is to be interpreted as an attempt to relieve intervertebral compression; when the curvature is towards the same side (ipsi- or homo- lateral) it is probably nerve-root tension which the body is trying to avoid. Another element of deformity usually present is elimination of lordosis; such flattening of the lumbar hollow may be so severe as to constitute a lumbar kyphos. These deformities which are fixed by muscle spasm are comparable to the hamstring spasm which fixes a deranged knee in flexion. A rarer anomaly is an alternating type of sciatic scoliosis (ischiatica alternans) in which the patient may vary the side of his displacement, sometimes voluntarily; at one moment ipsilateral, another time contralateral; but never can he hold the spine straight. Closer analysis of such a case reveals that the curves are not mirror images of each other. In one case reported by the writer (Capener, 1933) it was found that ipsilateral scoliosis had its apex at a higher level in the lumbar spine than the contralateral curvature.

I have often wondered whether herniation of the arachnoid farther into an intervertebral foramen occurs; possibly through local defect in its coverings or from frequent sudden rises of intrathecal cerebrospinal pressure. In one case of sciatica (Fig. 6, C), radiographed with lipiodol, I did find an unusually long prolongation around a nerve root beyond the confines of the intervertebral foramen. This, of course, need not be the explanation of the commonly observed remark of patients that sciatic pain is increased by sneezing or coughing. The bombardment of an oedematous nerve root by a sudden wave of increased intrathecal pressure is sufficient to account for it. Compression of the jugular veins will act in the same way: for example, with the patient sitting comfortably and the knee of the affected side extended to a point just short of that necessary to cause sharp sciatic pain, compress the jugulars—with intraspinal lesions the patient will *probably* experience sharp pain in the affected sciatic distribution. Even more significant is it when the test is carried out on the unaffected leg and pain is produced on the affected side. In such tests it must be remembered that straight leg raising (Laseague's test) necessarily causes elimination of the normal lumbar curve and produces all that goes with lumbar flexion. Laseague's test on the unaffected limb, if it gives pain on the affected side, is much more significant than when performed on the affected side alone. It does then indicate that pain is being produced by spinal movement, and not by stretching the affected sciatic nerve trunk.

The Ligaments

Two ligamentous structures in this region call for particular comment. The first is the ligamentum flavum, which has come into prominence recently because of the more frequent operations for sciatica. This " yellow " ligament is an interesting structure because, in a histological sense, it is composed of almost pure elastic tissue fibres and because a condition of " hypertrophy " has been described. It bridges the gap between each neural arch posteriorly; it thus completes the protection of the intraspinal tissues, and because of its density and elasticity acts like the flexible sheath to a motor brake-rod or carburettor control. At operation it is not infrequently found to be thickened, and appears to constrict the deeper tissues. Dockerty and Love (1940) have shown, however, that, rather than hypertrophy, the condition is the result of degeneration, and the thickening is due to invasion and replacement of elastic tissue by fibrous tissues. Probably it is secondary to local irritative changes—*e.g.* osteoarthritis or nuclear retropulsion. It is a convenient condition on which to pin a diagnosis when laminectomy is otherwise negative; but we deceive ourselves if we think that this is the real cause of the patient's trouble.

The *ilio-lumbar* ligaments provide an important means of anchoring the fifth lumbar vertebra. This vertebra has potentialities for considerable rotation, and is exposed to an amount of shearing stress not found elsewhere in the spine. Extending from the transverse processes (which are placed relatively more anteriorly) these ligaments (Fig. 3) are attached, at each side, to the crest of the ilium; are well placed for the control of rotation, and together provide a sling restraining forward movement of the last lumbar vertebra. That this is so is shown

decisively by some patients with spondylolisthesis, where the ligaments become calcified, as so often happens when ligamentous bands are exposed to long-standing tension of a pathological degree (e.g. the interspinous ligaments in tuberculous kyphosis). It requires little stretch of the imagination to envisage lesser degrees of ligamentous fibrosis as the result of trauma, such as, for example, occurs elsewhere in occupational lesions. It is worth noting, in passing, that from this ligament arises some of the fibres of the quadratus lumborum muscle.

Weight Transmission

So far I have discussed the vulnerability of the lumbo-sacral region mainly from the aspect of dynamics. There are, however, questions of statics which are no less important. Let us start first with the lumbo-sacral angle, which has been variously described by different authors. Most frequently it is used to denote the angle formed between the anterior surfaces of the last lumbar and first sacral bodies. Considered in terms of statics, the angle described by Whitman is more significant. According to him the lumbo-sacral angle is that between the upper surface of the sacrum and the horizontal. It matters little whether this angle is measured thus or in relation to the vertical. To avoid confusion I will use the term " lumbo-sacral inclination," which from a mechanical viewpoint is the angle to the horizontal of a plane midway through the lumbo-sacral intervertebral disc. There is no record that lumbo-sacral angles have ever been measured in living erect human beings: one suspects that measurements have been done on cadavers or in recumbency—both fallacious. I have radiographed the *lumbo-sacral inclination* in a number of standing individuals and have found the angle to be about 25–30 degrees (Fig. 7B). In the habitual lordotic person the angle is greater; in an individual with short hamstrings it is less. The angle is of course very variable according to posture. When the spine is hyperextended, or when the individual sits with knees extended, the inclination becomes almost horizontal; as it also does with Goldthwaite's back-flattening exercise. When standing with the spine and hips flexed, the lumbo-sacral intervertebral plane tilts forward to the vertical and beyond. In the joint between the fourth and fifth lumbar vertebra the plane of the intervertebral disc, except in lordotic people, is almost horizontal. The importance of the lumbo-sacral inclination is in the shearing stress exerted upon the intervertebral disc, and the consequent tendency for the whole lumbar spine to slide forwards off the sacrum. That this sliding does, to a certain degree, occur is seen in cases of *spondylolisthesis*, a condition which is far commoner than is usually thought. Mathematically, it can be shown that for an angle of 30 degrees the shearing force amounts to 43 per cent. of the superincumbent weight. The lumbo-sacral inclination lies a little above the centre of gravity in a standing person; therefore it can be estimated that if he weighs 140 lb. the shearing force will amount to about 27 lb.; which is much smaller than one would have thought (Fig. 7 B). Obviously in carrying weights the force will increase proportionately.

In an intact last lumbar vertebra the interlocking of its lower articular facets with those of the sacrum clearly counterbalance shear. The ilio-lumbar ligaments play a part, as does also the intervertebral disc. I am going into this in more detail because I believe that in the human machine and its disabilities not enough attention is directed to the internal stressing of its materials—in this region as important as anywhere. Of particular importance is the nucleus pulposus which though composed of soft tissues expands when released from its coverings; thus constituted it is clearly compressible. If in a lumbo-sacral skeleton one places a steel ball of the same size and in the same position as the nucleus pulposus, but embed it in rubber instead of the annulus, and then erect the specimen in a position comparable to that of erect posture with a superimposed weight, interesting information can be obtained. Briefly the ball sets up reactionary forces (Newton's Law) the effect of which is to " push "

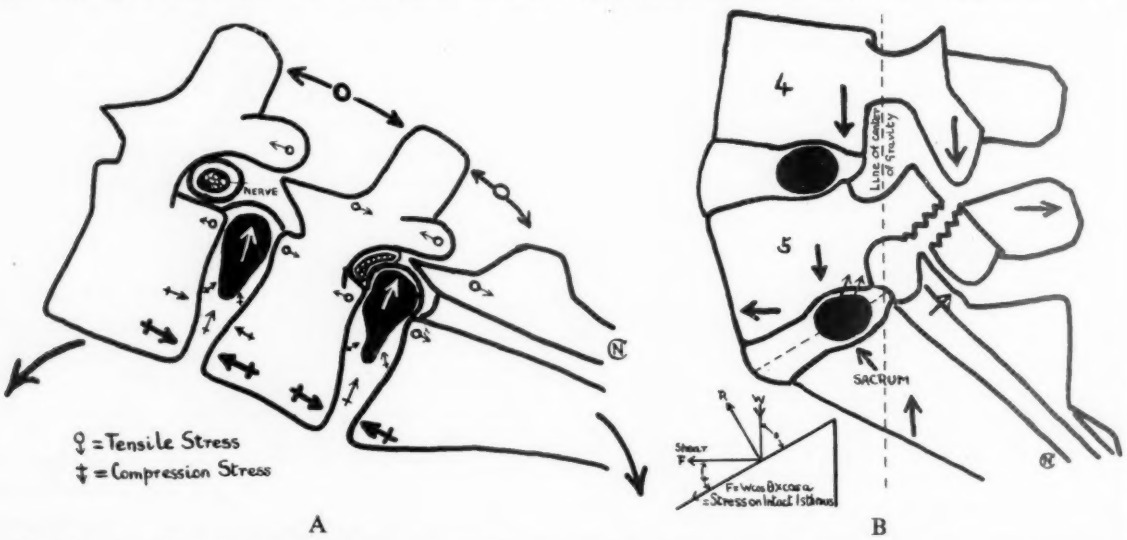


FIG. 7.—A.—The mechanics of nuclear retropulsion. B.—The mechanics of spondylolisthesis (the nucleus pulposus exerts reactionary force to shear).

the last lumbar vertebra backwards and upwards—in other words the ball tends to prevent it from sliding forwards. In the body, however, as the nucleus is a compressible structure its influence mechanically is even greater than the ball, because under weight it exposes a larger surface for the opposition of shearing force. In spondylolisthesis, where there is lack of continuity in the posterior neural arch between the upper and lower articular facets, the influence of restraint by both the ilio-lumbar ligaments and by the nucleus pulposus is probably much greater. Spondylolisthesis in its earlier state, that is *spondylolysis*, is probably present in about 5 per cent. of people, a frequency that adds point to discussions of lumbo-sacral vulnerability.

Lumbo-sacral Musculature

In 1934, before we heard much about the lesions of the intervertebral discs, I stated that I believed that 75 per cent. of the disturbance of lumbo-sacral sciatica was of a physiological nature—meaning muscle spasm. This may be an exaggeration; nevertheless I still believe it is near to the truth. The wrenched ligament, the retropulsed intervertebral nucleus, the stretched nerve root, whatever the inner lesion, is merely the trigger released; the explosion is in the muscular system. It may be large or small. What we later have to contend with is the atrophy and the dissociation of coordination in the muscles. Such atrophy, so obvious in the thigh muscles after derangement of the knee, is not so apparent in spinal lesions.

A simple picture of the posterior spinal musculature can be obtained if we regard it as comprising a continuous mass arising mainly from the sacrum and ilium on each side and passing upwards towards the neck, where it becomes more specialized. Throughout its course its deeper surface receives reinforcements from the bony structures upon which it lies. The superficial part of the mass splits into three perpendicular bundles attached respectively to the rib angles, the transverse and spinous processes. The deeper part of the muscle comprises shorter muscle units which have a more oblique course according to depth, but in the main they pass from transverse processes below, upwards and inwards to the spinous processes one or more vertebrae above, the more superficial passing over five or more vertebrae. The anterior spinal muscles are the abdominal muscles. The lateral spinal muscles comprise the extensions of the abdominal muscles into the loin, together with the psoas and a muscle which is often overlooked—namely, the *quadratus lumborum*. This arises from the iliac crest and ilio-lumbar ligament and passes upwards to be attached to the tips of the lumbar transverse processes and to the lowest rib. It is described as being a lateral flexor of the spine and a weak extensor. As a lateral flexor it is necessarily affected by the rotation (or torsion) that always accompanies side bending. The quadratus lumborum lies very close to the axis of backward and forward movement—so close, in fact, that in forward flexion it is possible for the muscle to act as a flexor. Normally spinal flexion rarely needs any flexor muscles; it is a gravity aided action which chiefly requires extensor muscles to control it. One can, without greatly stretching the imagination, appreciate circumstances in which, the "trigger" having gone off with the lumbar spine flexed, the quadratus lumborum is caught by explosive muscle spasm in a flexion phase. Should the spine also be twisted at the same time, it is possible for the quadratus on one side to be fixed in flexor spasm while the opposite is in extension. This is somewhat hypothetical, but I believe that in some such disorder is to be found one of the explanations of what, to some patients with an acute attack of lumbago, is an almost catastrophic disturbance. These muscles must, therefore, for coordinated action require a nice adjustment of control. Under conditions of fatigue or strain, physical or mental, this regulation may more readily break down. Is it too fanciful to regard minor degrees of such disturbance in other muscles as well, by causing atrophy, fibrosis, and adhesions, to be the same thing as much of the fibrositis about which we hear in sciatica?

It is worth considering the phases of muscular activity which have to be coordinated; thus can the complexity of the spinal problem be more fully appreciated. These are:—

A. *Simple Shortening Contraction.*—The "normal" anatomical action, *e.g.* of the biceps in flexing the elbow or the gluteals in extending the pelvis on the femur.

B. *Maintained Contraction* (*static*).—As when the quadriceps is tensed without movement of the knee.

C. *Lengthening Relaxation.*—As when the triceps elongates to permit the biceps to flex the elbow.

D. *Lengthening Contraction.*—The converse to A and seen when a weight in the hand is lowered by extension of the elbow. This is the "paradoxical" action of the biceps. It is the same action that the spinal extensors have in controlling flexion of the spine and in the quadriceps particularly in walking down hill.

E. *Maintained Relaxation.*—The static condition of complete relaxation.

F. *Shortening Relaxation.*—The converse to C, as when the triceps shortens during the paradoxical action of the biceps in controlling elbow extension against gravity. Similarly in the spinal flexors when the extensors are in action against flexion of the spine.

Summary

The lumbar spine presents many features in anatomy and mechanics the appreciation of which should help in the elucidation of the clinical problems of sciatica. It remains to state that in clinical investigations more note should be taken of such factors.

The history should have regard for details of trauma, particularly of the more slowly operative or static types. The habitual postures of occupation

should be inquired into as well as the posture assumed during any isolated incident which has precipitated an acute sciatic attack. Such an inquiry may be found just as important as the well-recognized details of the mechanics of knee-cartilage injuries. Additional information should be sought about those postures which either aggravate or relieve symptoms—matters which will be available for corroboration during the physical examination. During the latter more attention than is usual should be paid to the influences of spinal movement upon the intraspinal and intervertebral tissues and to the changes in intrathecal pressure. Finally, we should make a closer study of the muscular control of the lumbar region.

In this paper I have presented information and a few theories about the mechanics of the lumbo-sacral region. Amongst the points I have discussed from a new angle are the rotation of the lumbar spine, the movements of the meninges and nerve roots, the mechanics of the nucleus pulposus, weight transmission and matters relating to the " internal stresses of materials," and the action of the quadratus lumborum muscles. In the course of the paper it has been necessary to touch on some very elementary facts, but the whole is given as a guide to the investigation and elucidation of the problems of sciatica for clinical workers.

The researches upon which this is a brief report started in the departments of anatomy, radiology, and orthopaedics at the University of Michigan and have been continued during the past twelve years in the orthopaedic hospitals of Devon. It owes much to the inspiration of the late Dr. P. M. Hickey, Professor of Roentgenology at the University of Michigan and to Sir Arthur Keith. To Professor F. H. Newman of the Department of Physics, University College of the South West, Exeter, I am most grateful for his guidance upon the mechanical problems involved. For the radiological studies upon normal spines in standing and other postures, I am indebted to the patience of various past and present members of my Staff in the Princess Elizabeth Orthopaedic Hospital, Exeter.

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EXPERIMENTAL TRANSMISSION OF RHEUMATIC FEVER

BY

W. S. C. COPEMAN

The opportunity arose in 1942 of obtaining certain adult volunteers for an experiment on the transmission of rheumatic fever in blood. This experiment will be recorded in full, although it is realized that owing to its limited scope it is not of great scientific value. It is, however, believed to be the only one of its type on record, and it is therefore of interest and seems to give experimental confirmation to certain hypotheses formulated in an article in the last issue of this journal. It was undertaken in association with Prof. Paul Beeson (then of the Harvard Field Hospital Unit), and was cut short by the exigencies of war service. Briefly it consisted in injecting the blood of a patient suffering with typical rheumatic fever into five volunteers. The blood of those who showed any reaction was then also taken and injected into a further group of volunteers; and so on. It was only in the fourth " generation " of volunteers that no further reaction of any sort was noted.

SOURCE OF INFECTION

The original donor case of rheumatic fever was a man (A. D.), aged 24, married, with no children. Father had rheumatism which started in childhood. Patient had all childish diseases. Pleurisy at the age of 12, which kept him in bed for several weeks on suspicion of tuberculosis. In Army bakery 2 years. No rheumatism. Quinsy, 2 months and again 2 weeks previously to onset of present illness. On Dec. 20 he awoke with acute pain on outer aspect of right knee. This pain was cramp-like, and he had to flex and immobilize it to get any relief. Poulticing and painting with iodine also gave relief. Next day he was able to walk about, although he noticed that both knees were swollen. He slept well, but next morning his knees were very stiff, rather painful, and more swollen. In addition he had pains elsewhere, chiefly round the shoulders. Given light duty and lotion.

On fourth day the pains were all much worse, and the knees so swollen and painful that he could not get up. Towards evening he seemed ill, was sweating profusely, and had a temperature of 103° F. He was then admitted to hospital. Next morning he still felt very ill and complained of headache and pains round the shoulders, and inability to move either knee owing to pain and swelling. He was still sweating profusely, but his temperature was only 100° F. All the muscles of the lower limbs seemed tender and the first cardiac sound was muffled. The spleen and liver were not palpable. The urine was very acid. The B.S.R. was 40 mm. (Westergren). Red blood count, normal. W.B.C., 8175 per c.mm. A sample of blood was taken for the purpose of the experiment. Next day his condition was similar, but on the following day the pains had disappeared and the temperature fell to normal and severe labial herpes developed. He made a good recovery, which was interrupted by an attack of bronchitis from which he took some time to recover, and during which the pains returned mildly. Before discharge he complained of lumbar pain, found to be due to " myalgic spots " at the borders of the lumbar muscles. A faint systolic murmur which had appeared at apex during the first week after admission was still present, and he complained of some dyspnoea on moderate exertion.

A month after the onset, when the temperature had been normal for about 20 days but the B.S.R. was 19 mm., a further specimen of blood was taken and injected into 5 other volunteers, with no result whatever.

CLINICAL OBSERVATIONS ON VOLUNTEERS

Dec. 28, 1941.—Blood from patient A. D. taken at 9.15 a.m. and injected intravenously at 2.15 p.m. to the following five persons, each of whom received 5 c.cm.: D. F., G. I., J. L., and 2 others.

Dec. 31.—D. F. complained of pain in the left shoulder two days after injection; no other complaints. A tender spot could be palpated along the upper border of the left trapezius. On the afternoon of Dec. 29, G. I. complained of not feeling well; temperature was 102° F. (axilla). At 5.30 p.m., put to bed; complained of headache. That night there was definite pain on moving the right arm. Temperature on morning of Dec. 30 was 99·4° F. (axilla) but normal that evening. On examination the patient appears " washed out." Complained of a pain at the upper part of the back of the neck, but no definite area of tenderness found there. Also complained of pain in the region of the right triceps brachialis and in the right lumbar muscles. No sharp localization could be detected. On Dec. 30 at 3.30 p.m. J. L. complained of headache. T=99·8° F. (axilla); no other symptoms. On Dec. 31 temperature was 100° F. (axilla). On physical examination a tender spot was found in the right trapezius about the mid-line; no nodule felt. Patient had no difficulty in moving arms or turning head. Defibrinated blood from G. I., and J. L., pooled and inoculated intravenously as follows: M. O. and 3 others, 4 c.cm.

Jan. 4, 1942.—G. I. still appears ill. Temperature this morning 99° F. (axilla). Complained of pain in right lumbar region near the spine, back of the neck near the insertion of the trapezii into the occiput, and right arm region of deltoid muscle. Tenderness difficult to estimate; no spasm found. Blood taken for B.S.R. (50 mm.) and culture; 25 c.cm. defibrinated for further transfer. J. L.: No more fever, no complaints, no tenderness, discharged. D. F.: No more pain in left shoulder; still slight tenderness in the left trapezius. M. O.: No symptoms or signs now, but states " a day or two ago " she had fairly severe pain in left side of the neck region and upper portion of the trapezius. No tenderness there now. The three others had no signs or symptoms following inoculations made with fresh defibrinated G. I., J. L., blood except that in one of them temperature rose to 100° F. at night. No complaint of pain, and no subsequent symptoms.

P. R. and three others each received 4 c.cm. intravenously from G. I.

Jan. 11.—G. I. appears better, but is weak and shaky; still in bed; temperature normal for past week. Still complains of pain in three regions mentioned previously; no tender spot detected. P. R. on Jan. 6, complained of pain in region of insertion of left deltoid. Next day complained of pain in arm and left side of back of neck (trapezius medial to left scapula). On Jan. 8, had pain in both lumbar muscle groups and left upper gluteal region. On Jan. 9, complained of pain in lumbar and gluteal regions and in left chest, about the sixth interspace, lateral

aspect. To-day she looks miserable, and there is marked tenderness in the above-mentioned areas. The thoracic area is particularly tender. She has never had any elevation of temperature. Blood taken for culture, B.S.R., etc., and also defibrinated and 4 c.cm. inoculated intravenously into S. U. and three others.

Jan. 12.—S. U. had temperature of 99·8° F. (axilla) in morning, 97° F. in evening. Complained of pain in right arm, below elbow. Jan. 13: T.=99·2° F. in morning, 97·6° F. in evening. Jan. 14: T.=99·6° F. in morning, 97° F. in evening. Jan. 15: T.=98·8° F. in morning. Patient examined; no specific complaint obtainable; no tender area found. Blood taken for usual studies and for transfer. G. I.: No fever, no complaints, no physical signs. P. R.: Patient better, but still complains of pain in lower part of back; still has some tenderness in lumbar region, and over lower ribs bilaterally.

Jan. 16.—About Jan. 7 Nurse H., who was attending these patients, wakened in night with severe pains in legs, above, below, and in the knees. Movement was painful. Pain extended up and down both lower extremities, even involving both gluteal regions. There was one especially tender spot on the right thigh, three inches above the knee. The pain seemed worse when lying still, and she could get no rest in bed. She sat up most of three nights. She vomited six times in four days. No numbness or tingling. No pain above the hips. She had no respiratory symptoms. No feverish or chilly sensations. No headache.

Jan. 16.—3 c.c. defibrinated blood (3 c.cm.) from S.U. inoculated into 4 other volunteers with no result.

Jan. 22.—Nurse H. still has pains in backs of legs. For three days last week she had pain and stiffness in back of neck (both trapezii), with limitation of movement. She still has trouble in sleeping because of the pain. No fever or headache. G. I.: Up last week, no pain. P. R.: About the same. Still tender in previously described areas. To be allowed up in 2 days. Developed some pain on getting up, and on Feb. 2nd was reported as ? chronic fibrositis. S. U.: Still in bed, no fever, no apparent pain, but ? slight systolic murmur, apex.

Summary

From the results of this experiment (which it is realized is incomplete and insufficiently controlled) it would look as though an infective agent was present in the blood of the original patient (A. D.) with rheumatic fever, and that this was transferred on the fourth day of his illness to the volunteers, producing mild attacks of rheumatic fever in two of them (G. I. and J. L.) after 24 hours, and pain in the shoulder muscles without pyrexia—fibrositis—three days later in another (D. F.).

The blood from these first two cases was pooled on the third day and again passed on, producing what appeared to be an attack of a febrile fibrositis on the second day in one of four recipients (M. O.).

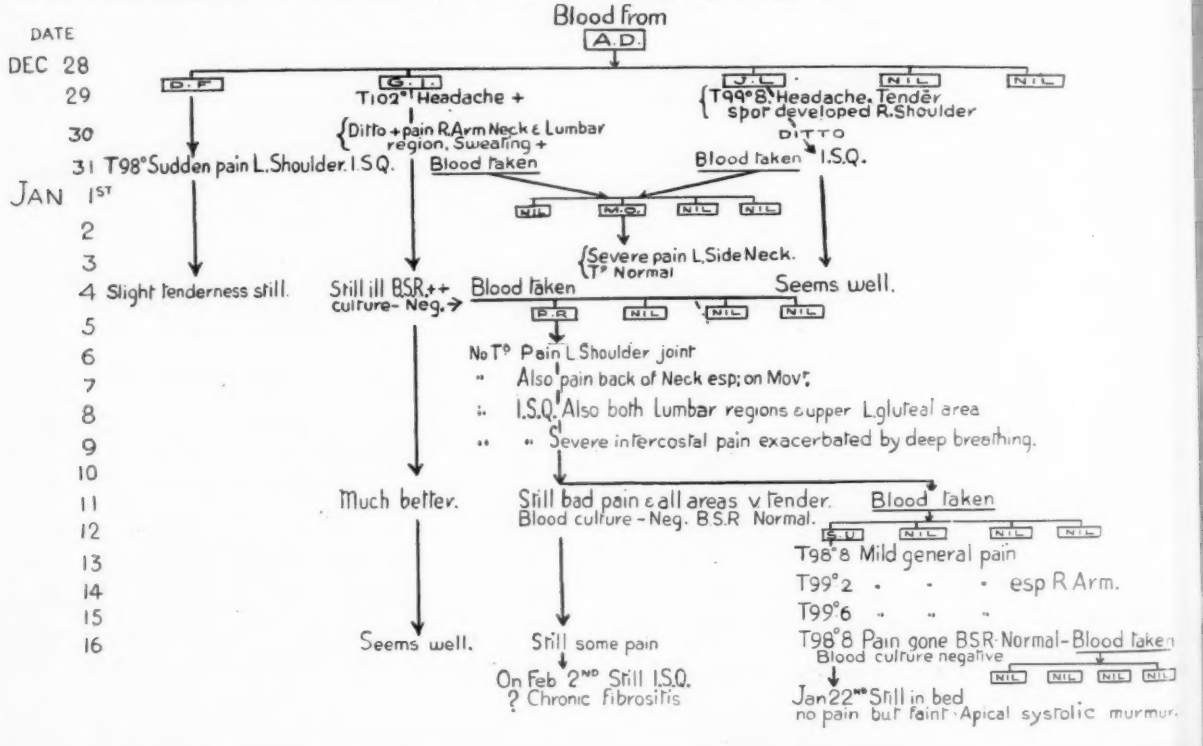
The blood of the more severely affected of the first two cases (G. I.) was again transferred, on the seventh day of his illness, to four other volunteers, one of whom (P. R.) developed a painful generalized fibrositis which six weeks later seemed likely to become chronic.

The blood of this patient (P. R.) had been transferred to four recipients on the seventh day after he himself had received his blood infection. One of these developed mild fever and generalized pains which lasted a few days only, but five weeks later a faint apical systolic murmur was heard. Blood taken from this patient on the fifth day produced no effect on four recipients.

The attack of rheumatism suffered by the nurse in attendance may have been a coincidence.

Conclusion

It might be surmised from this experiment (1) that rheumatic fever can be transmitted in the blood of an active sufferer, and (2) that the syndrome of rheumatic fever may be a synthesis of two elements, (a) a febrile non-specific element, and (b) a specific fibro-toxic (and cardiotoxic) element; since it seemed that there was some clinical evidence of these two elements developing independently in certain of the volunteers.



THE MICRO-DETERMINATION OF GOLD IN BIOLOGICAL FLUIDS AND TISSUES

BY

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The increasing interest in the use of gold salts as chemotherapeutic agents in the treatment of certain diseases (Hartfall, *et al.*, 1937) has made apparent the necessity of an accurate, specific, and sensitive method for the determination of gold in various biological materials. Moreover, the lack of information regarding the metabolism and pharmacology of this element further emphasizes this need. Previous methods have employed either an electrolytic precipitation, a colorimetric reaction, or a titrimetric procedure. The electrolytic methods such as those of Cadwell and Leavell (1919), and Lombolt (1926), involve too many technical difficulties to be of practical value. The numerous colorimetric methods (Stanbury, 1932; Leulier, Bernard, and Loisy, 1937) lack both specificity and accuracy. The method of Merejkovsky (1933) seems to be specific and is probably the best, but the large blank from the reagent itself and the inadequate directions of the author make it impossible to duplicate the original results. The iodometric titration first employed by Peterson (1898) and modified most recently by Hansborg (1925) cannot be applied to tissues and blood because the ferric iron present in these substances interferes with the reaction. This difficulty has been overcome by Tukats (1933) by a preliminary precipitation of the gold with oxalic acid. However, this preliminary procedure makes it impossible to apply the method to the determination of very small quantities of gold. Pollard (1937) determined gold after conversion to the chloride by titration with hydroquinone using o-dianisidine as an indicator. In order to secure quantitative results it was necessary for other inorganic salts to be absent. This condition was obtained by a preliminary precipitation of the gold from the original substance by use of a hydrochloric acid solution of tellurium saturated with sulphur trioxide. A modification of this method for use with urine (Jamieson and Watson, 1938) required large volumes of urine and was limited in sensitivity.

Because none of these methods are entirely adequate, it was thought it would be of value to develop an accurate and specific modification of Pollard's (1937) procedure for the quantitative determination of gold in small amounts of blood, urine, and tissue.

Principle of Present Method

The present method involves the determination of gold by the use of the colour reaction produced by dianisidine and auric chloride in a slightly acid solution buffered with potassium bifluoride. This colour production follows Beer's law and can be quantitatively measured in an Evelyn photoelectric colorimeter. Preliminary digestion by use of sulphuric acid and hydrogen peroxide is necessary to oxidize all interfering organic substances. Since the colour reaction is specific for the chloride or bromide of gold, it is necessary to convert the separated gold to the chloride by aqua regia. All excess sulphuric acid must be previously removed by evaporation to absolute dryness in order to secure a quantitative conversion of gold to the chloride. Likewise after conversion to the chloride, excess aqua regia must be removed because nitrosyl chloride as well as nitric acid produces a colour with dianisidine. Further, the presence of any excess acid will inhibit the production of the final colour with gold chloride. Potassium fluoride and hydrochloric acid are then added to the solution to produce the buffered acid solution. In addition, the fluoride is necessary to prevent the reaction of any ferric iron present with the dianisidine (Kul'berg, 1936).

Experimental

REAGENTS

Standard gold solution.—199·84 mg. of gold chloride ($AuCl_3.HCl.3H_2O$ (Merck and Company)) are transferred to a 1000-c.cm. volumetric flask and diluted to volume with distilled water. 1 c.cm. of this solution is equivalent to 0·1 mg. of gold. This stock standard is stable for two to three days.

Potassium fluoride.—10 g. of this salt are weighed out accurately and transferred to a 100-c.cm. volumetric flask and diluted to volume with distilled water. After the salt dissolves, the solution is filtered. This solution is stable for two days.

Potassium bifluoride.—(Merck and Company). 0·75 g. of potassium bifluoride are weighed accurately and trans-

* The Rackham Arthritis Research Unit is supported by the Horace H. Rackham School of Graduate Studies of the University of Michigan.

ferred quantitatively to a 100-c.cm. volumetric flask and diluted to volume with distilled water. This solution should be made fresh every 12 hours.

Dianisidine.—(Technical. Eastman Kodak Company). 250 mg. of dianisidine are weighed accurately, transferred to a 250-c.cm. volumetric flask, and 150 c.cm. of water and 1 c.cm. of concentrated hydrochloric acid are added. The solution is then diluted to volume with distilled water and will remain stable for one day.

Hydrochloric acid (1: 4).—25 c.cm. of concentrated hydrochloric acid are transferred to a 100-c.cm. volumetric flask and diluted to volume with distilled water.

Hydrochloric acid (1 : 12).—41·5 c.cm. of concentrated hydrochloric acid are transferred to a 500-c.cm. volumetric flask and diluted to volume.

Aqua regia.—10 c.cm. of concentrated nitric acid are added to 30 c.cm. of concentrated hydrochloric acid. This solution should be made every day.

PROCEDURE

A known amount of urine (10 to 50 c.cm.) is placed in a Kjeldahl flask * which has been graduated to 100 c.cm. To this flask 3 to 5 c.cm. of concentrated sulphuric acid are added. A small glass bead and several drops of caprylic alcohol are introduced into the mixture, which is then slowly heated on a Kjeldahl digestion rack until the solution begins to char. The heating is continued for 15 to 20 minutes, after which the flask is cooled and a 30 per cent. solution of hydrogen peroxide (superoxol) is added drop by drop until the solution is water clear. This requires approximately 15 to 20 drops. The solution is then evaporated to complete dryness over a micro-burner. The sulphuric acid fumes are removed by inserting into the neck of the flask a glass tube which is connected through a water trap to a water pump. The flask is cooled, 2 c.cm. of aqua regia are added, and the flask is heated on a micro-burner until the solution is evaporated to 0·2 to 0·3 c.cm. The flask is removed, and while still warm a slow stream of air is introduced into the flask, which evaporates the remaining acid and moisture. During the heating the flask should be 5 to 6 cm. above the tip of the flame to avoid local overheating, which will decompose the gold chloride. After cooling, the precipitated inorganic salts are redissolved by addition of about 75 c.cm. of distilled water. 0·75 c.cm. of hydrochloric acid (1 : 4), 8 c.cm. of potassium fluoride, and 1 c.cm. of dianisidine solution are added to the flask. The volume is made up to 100 c.cm. with distilled water, the flask stoppered, and the solution thoroughly mixed. The solution is then transferred to a colorimeter absorption tube, the tube being filled to within 1 to 2 cm. of the top. After stoppering, the pink colour, which reaches its maximum intensity within 5 to 10 minutes, is read in the Evelyn photoelectric colorimeter, using filter number 440 with a centre setting on the galvanometer of 76. The stoppering of the graduated Kjeldahl flask and absorption tube is necessary to prevent a slight colour development of the reagents in the presence of air.

The same procedure can be applied to blood, using 1 to 5 c.cm. of plasma; this is placed in a micro-Kjeldahl digestion tube which has been graduated to a 15 c.cm. volume. The initial digestion requires 1 c.cm. of sulphuric acid, and the conversion to the chloride is accomplished by 0·5 c.cm. of aqua regia. The colour is then developed by the addition of 0·1 c.cm. of hydrochloric acid (1 : 12), 1 c.cm. of potassium bifluoride, and 0·4 c.cm. of dianisidine. This is then diluted to 15 c.cm. graduation and the colour read as in the case of the urine. Stoppering of the Kjeldahl flask and absorption tube is unnecessary because the potassium bifluoride prevents the production of any colour of the reagents in the presence of air.

The tissue (0·5 to 10 g.) to be analysed is placed in an accurately calibrated 100-c.cm. Kjeldahl flask whose neck has been shortened to 5 or 6 cm. 10 to 20 c.cm. of concentrated nitric acid and a few drops of caprylic alcohol are added and the tissue allowed to stand for a few hours until solution has occurred. 10 to 15 c.cm. of concentrated sulphuric acid are introduced into the flask, which then is heated on a Kjeldahl digestion rack until the tissue is thoroughly charred. After the digest is cooled, a 30 per cent. solution of hydrogen peroxide (superoxol) is added drop by drop until the solution is water clear. The solution is evaporated to dryness over a micro-burner. The sulphuric acid fumes are removed by inserting into the neck of the flask a glass tube which is connected through a water trap to a water pump. The contents of the flask are then treated with aqua regia to convert the gold to the chloride. If the tissues contain amounts of gold less than 200 micrograms, the colour is developed and read as previously described directly with the entire contents of the digestion flank. However, if larger amounts of gold are present the aqua regia solution in the flask is heated to boiling, cooled, and transferred to a 100-c.cm. volumetric flask. The Kjeldahl flask is rinsed twice with 15 to 20 c.cm. of boiling aqua regia, and the washings are added to the volumetric flask, which is made to volume with distilled water. Aliquots containing 50 to 150 micrograms of gold are transferred to the shortened graduated 100-c.cm. Kjeldahl flasks, evaporated to dryness, remaining traces of sulphuric acid being removed as before, and the gold reconverted to auric chloride by boiling with aqua regia. The aqua regia solution of this aliquot is then used to develop the colour. Any fatty or muscular tissue (legs, gastro-intestinal tract) is treated in the same fashion, except that the digestion is carried out in a 300-c.cm. Kjeldahl flask with the addition of selenium as a catalyst. After evaporation to dryness with complete removal of the sulphuric acid, the residue is put into solution by boiling with three 40-c.cm. portions of aqua regia. All the washings are collected in a 250-c.cm. volumetric flask, made to volume with water, and aliquots containing 50 to 150 micrograms of gold transferred to the special 100 c.cm. Kjeldahl flasks. The procedure is then carried out as described above for urine.

This same procedure can be applied to large organs or carcasses of small experimental animals as well as to dried animal and human faeces, the only modification being that a 500-c.cm. Kjeldahl digestion flask is used.

Special attention should be called to the amount of 1 : 4 hydrochloric acid added in the colour reaction, for the rate of colour development and fading depends upon the ratio of fluoride to acid. The amount of acid which remains in the flasks after conversion with aqua regia depends upon the amount of inorganic salts present. A maximum colour will develop in 6 to 8 minutes if 0·75 c.cm. of 1 : 4 hydrochloric acid is used in direct determination of gold in tissues or urine. In determinations of gold in carcases or faeces, 1·25 c.cm. of the acid must be added and 2·5 c.cm. in the case of diluted tissue digests. It is best to read the colour at one-minute intervals during the development until the maximum colour is reached and fading begins. Since it requires only 15 seconds to make a reading, four tubes may be read easily at minute intervals.

* 100-c.cm. Kjeldahl flasks with necks shortened to 506 cm. are the size used, as when they are accurately calibrated with a Bureau of Standards 100-c.c. pipette the calibration mark falls in the narrow neck portion of the flask.

Results obtained in Tests with Pure Solutions of Gold Chloride

A series of determinations were conducted using varying quantities of gold ranging from 5 to 300 micrograms. The results are presented in Table 1. When the galvanometer was adjusted to 100, a stoppered tube containing an aliquot of the blank solution made up of 3 c.cm. of hydrochloric acid (1:4), 8 c.cm. of potassium fluoride, and 1 c.cm. of dianisidine diluted to 100 c.cm. volume, a centre setting of 76[1] was obtained as compared to 76[0] when water alone was used. The ratio of photometric density (" L values ") to the concentration of the test solution is a constant (K, Table 1). Reproducibility of results in these tests was excellent. Below the quantity of 5 micrograms of gold there is a significant deviation from the " K value." However, this can be overcome in unknowns by using sufficiently large samples of the substance to give readings in the range of 5 to 300 micrograms, which was the range desired in urine. For blood, however, the desirable range was 5 to 30 micrograms. The blank solution for blood which was diluted to 15 c.cm. contained 0·1 c.cm. of hydrochloric acid (1: 12), 1 c.cm. of potassium bifluoride, and 0·4 c.cm. of dianisidine solution, gave a centre setting of 76[0]. The " K value " determined for the range (5 to 30 micrograms) was constant, averaging 0·020 (0·019 to 0·022).

TABLE 1

Reproducibility of the Chemical Method for the Determination of Gold Chloride and the Constancy of the Ratio of Photometric Density to Concentration of the Compound.

Gold present	Number of tests	Range of galvanometer readings [1]	Average photometric density [2] (" L values ")	Calculated K value [3]
Micrograms				
5	3	96^2, 96^3	0·0155	0·0031
10	4	93^2, 93^3	0·0281	0·0030
15	5	89^3, 90^0	0·0470	0·0031
20	5	87^0, 87^1	0·0605	0·0030
25	5	84^0, 84^2	0·0757	0·0030
30	2	81^0, 81^2	0·0915	0·0031
50	4	69^2, 70^0	0·1580	0·0032
100	4	48^0, 48^2	0·3190	0·0032
150	3	32^2, 33^0	0·4850	0·0032
200	2	22^3, 23^0	0·6430	0·0032
250	3	15^3, 16^0	0·8030	0·0032
300	3	12^0, 12^1	0·9210	0·0031
			Average	0·0031

[1] These values correspond to the maximal pink colour produced when taken with a centre setting of 76^1.
[2] This is analogous to optical density as measured on a spectrophotometer and corresponds to the quantity (2 − log 10 of the galvanometer reading).
[3] These values are obtained by dividing the photometric density by the amount of the gold in the test solution.

Recovery of Gold from Urine, Blood Plasma, and Tissue

The results obtained by the addition of known quantities of gold chloride to various volumes of urine and blood plasma as well as to various tissues are summarized in Tables 2 and 3. In the case of urine, the percentage of recovery varied from 94·2 to 100. The dilution of the urinary digest overcomes any inhibitory effect of inorganic salts on the development of the colour as found by Pollard (1937). This is well illustrated by our results where the percentage of recovery is approximately the same for 50 c.cm. of urine as in 10 c.cm., although the concentration of inorganic salts is 5 times as great in the former. In blood, however, the error was slightly greater with the range of the percentage recovery being 84·2 to 100. The greatest error in the

TABLE 2

Recovery of known amounts of Gold Chloride added to Urine and Plasma

Material used	Volume	Gold added	Colorimeter reading	Gold found	
	c.cm.	micrograms		micrograms	per cent.
Urine	10	0	76^0	0·0	0·0
,,	10	20	86^3	19·90	99·5
,,	10	50	70^0	49·97	99·9
,,	10	150	34^1	150·00	100·0
,,	10	200	25^0	194·20	97·1
,,	10	250	17^3	242·26	96·9
,,	50	0	76^1	0·0	0·0
,,	50	10	93^2	9·42	94·2
,,	50	15	90^0	14·77	98·5
,,	50	20	86^3	19·90	99·5
,,	50	25	84^0	24·42	97·6
,,	50	50	70^2	48·97	97·9
,,	50	100	49^0	100·00	100·0
Plasma	1	0	76^0	0·0	0·0
,,	1	3	89^0	2·53	84·2
,,	1	5	81^3	4·38	87·6
,,	1	7	75^1	6·18	88·3
,,	1	8	71^1	7·11	88·9
,,	1	10	63^1	9·95	99·5
,,	1	20	40^0	20·00	900·0

TABLE 3

Recovery of Known Amounts of Gold Added to Tissues and Excreta of the White Rat

Material used	Gold added	Gold found	
	mg.	mg.	per cent.
Heart ..	0·0	0·0	0·0
	0·010	0·00895	89·5
	0·020	0·01946	97·3
	0·030	0·02968	98·9
Lung ..	0·0	0·0	0·0
	0·010	0·00928	92·8
	0·020	0·01973	98·7
	0·150	0·1471	98·1
Spleen ..	0·0	0·0	0·0
	0·020	0·0199	99·5
	0·100	0·100	100·0
	0·200	0·1916	95·8
Liver ..	0·0	0·0	0·0
	0·200	0·199	99·0
	1·000	0·971	97·1
	5·000	4·855	97·1
Kidney ..	0·0	0·0	0·0
	0·100	0·958	95·8
	2·000	1·974	98·7
	5·000	4·855	97·1
Whole legs	0·0	0·0	0·0
	0·500	0·4683	93·7
	5·000	4·797	95·9
	20·000	18·864	94·3
Entire carcase.	0·0	0·0	0·0
	2·000	1·829	91·5
	10·000	9·870	98·7
	40·000	39·420	98·5
Urine [1] ..	0·0	0·0	0·0
	0·300	0·294	98·0
	5·000	4·813	96·2
	10,000	10·000	100·0
Faeces [1] ..	0·0	0·0	0·0
	0·500	0·4064	81·3
	2·000	1·987	99·3
	5·000	4·920	98·4

[1] Gold added to urine and faeces collected over a two-week period.

case of plasma occurs in those instances in which less than 5 micrograms of gold are determined. For amounts greater than this there is essentially a 90 per cent. recovery. If a greater accuracy than 90 per cent. is desirable, a larger volume of plasma may be used. In tissues the recoveries vary from 90 to 100 per cent. except in one case in which 0·5 mg. of gold was added to faeces. In this instance recovery was 81·3 per cent. This comparatively low recovery is probably associated with the inhibition of colour development due to the presence of large amounts of inorganic salts in relation to the small quantity of added gold.

The specificity of the method in both urine and blood is shown by the colorimeter reading of 76 (Table 2) secured for digests of both urine and plasma. This value is identical to that secured with reagents alone. The same specificity is shown in the case of tissues where no gold was found when none was added. These results indicate that the intensity of the colour secured with these materials when they contain gold is due entirely to the presence of the gold and cannot be attributed to the reaction of the dye with any other inorganic salt.

Summary

An accurate and specific microcolorimetric method for the determination of gold by the use of the Evelyn photoelectric colorimeter has been described. The method is based upon the production of a stable red colour by the reaction between auric chloride and dianisidine in an acid medium in the presence of potassium fluoride. The method is applied to the determination of gold in urine and tissues in the range of 5 to 300 micrograms, and to blood plasma in the range of 5 to 30 micrograms.

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DETERMINATION OF BLOOD LEVEL OF VITAMIN C IN CHRONIC RHEUMATIC DISEASE

BY

ENID A. M. BRADFORD

In juvenile rheumatism and rheumatoid arthritis, and in other infective conditions, it has been shown that vitamin-C levels are lower than in controls receiving the same intakes of the vitamin (Harris, 1937; and Rinehart, 1935, 1936, 1937). The extent of the fall in these rheumatic conditions is not so pronounced, however, as in certain other infective states, notably in acute pulmonary tuberculosis. Recent work of Mouriquand *et al.* (1938) in France and Kodicek and Murray (1943) at Cambridge on guinea-pigs suggests that there may be some hitherto unsuspected connection between abnormal bone growth and vitamin-C intake. The two last-named workers found that guinea-pigs on a prolonged partial deficiency of vitamin C (which was not severe enough to cause any symptoms of frank scurvy) showed a tendency to ankylotic change in the knee-joints after very slight injury. A minute trauma, such as would occur in merely handling the animals for taking x-ray records, was sufficient to set in motion a hyperplasia of connective tissue in the haemorrhagic area of the injury, which led to immobility and ankylosis of the joint. This effect was independent of vitamin D.

With these facts in view it seemed worth while to explore the hypothesis that a prolonged partial deficiency of vitamin C may be one of the possible factors involved in the genesis of rheumatic arthritis. The work of Bourne on repair of bone in vitamin-C deficiency (1942) is also interesting in this connection. In view of this experimental work on animals it is possible that some mild forms of pre-clinical scurvy may masquerade as rheumatism, since the symptoms of early scurvy in the adult human are weakness and pains in the limbs with subsequent skeletal lesions at the junction of bone and cartilage, loosening of the teeth, etc.

From the work of Daniels and Everson (1936) and Keith and Hickman (1938) it is known that the administration of aspirin lowers the renal threshold of vitamin-C excretion, and practically all cases of arthritis take quantities of this drug to obtain relief. The successful control of rheumatic conditions may well require, among other factors, an adequate supply of vitamin C, and it is apparent that a wide field for research is open on this subject. Although clinical impressions are always open to uncertainty these results demonstrate the need for further observations on a statistical basis. In view of the practical difficulties of the usual saturation tests for patients attending an out-patient clinic the state of vitamin-C nutrition has been investigated by determining the fasting blood level. Prunty and Vass (1943) have recently shown that this gives reliable information compared with urine saturation tests.

Method

The method of Deeny, Murdock, and Rogan (1942) was used with some minor modifications of technique necessary for pathological specimens. The object was to find a rapid method which would indicate the circulating level of vitamin C, and hence whole blood was used. This was thought preferable to plasma, which is more easily influenced by recent meals, or to white corpuscles, which present complications in the technique of separation from other constituents of blood, and of obtaining comparative results. Many of the blood samples examined caused trouble due to frothing in the preliminary treatment with coal gas, and also in obtaining a crystal-clear final solution for titration. These difficulties were largely overcome by reducing the initial gas treatment to five minutes before diluting with water, and prolonging the second gas treatment to ten minutes. The total exposure to coal gas was thus the same as in the original method. All samples were analysed within an hour of withdrawal. It is important that the metaphosphoric acid should be freshly prepared each day to ensure efficient precipitation of the proteins.

Titrations were carried out in small, straight-sided Jena glass test-tubes measuring 85 mm.×10 mm. (Behring Venule tubes). With practice one tube can be used as control for the other duplicate by titrating against a white ground, which avoids the inaccuracy due to slight opalescence in the supernatant liquid from some pathological bloods. With these precautions an accuracy of 0·1 mg. per 100 ml. blood is easily possible, and has been checked by comparative determinations.

Results of Investigation

There is some uncertainty as to standards for vitamin C in blood. Since, however, according to various American workers (Abt *et al.*, 1936; Faulkner, 1938; Wright, 1938; Goldsmith, 1939 and Todhunter, 1940) a normal level is considered to be from 1·0 to 1·2 mg. per 100 ml. of blood, and

values between 0·5 to 1·0 mg. are taken by them to indicate a pre-scorbutic state, samples below 0·5 mg. per 100 ml. may be regarded for the present purposes, and without actual commitment as to the desirable levels, as " below the U.S.A. standard." The cases—Table 1A—were taken at random during March and February, but as our experience grew a certain amount of selection was made in the light of the symptoms of patients showing low vitamin-C levels.

TABLE 1A

Vitamin C in Blood of Rheumatic Patients

Month	Total No. of Cases	No. of Cases Showing a Blood Vitamin C of:		
		0·5 mg. per 100 ml. or below	0·5 to 1·0 mg. p. 100 per ml.	above 1·0 mg. per 100 ml.
Feb. ..	3	1	1	1
March ..	31	11	15	5
Apr. ..	26	18	5	3
May ..	26	18	7	1
June ..	54	29	17	8 (5 taking vit. C)
July ..	9	5	2	2
Aug. ..	4	1	3	0
Sept. ..	14	6	5	3
Oct. ..	10	5	2	3
Nov. ..	6	3	2	1 (taking vit. C)
Total ..	183	97	59	27
Percentage of total		53·0	32·2	14·8

TABLE 1B

Blood Vitamin C in Normal Controls and Vegetarian Blood

April ..	1	0	1	0
May ..	3	0	3	0
June ..	2	0	2	0
July ..	4	0	3	1
July Vegetarians	6	0	0	6
Totals	16	0	9	7

The figures in Table 1A indicate that a large proportion of the patients attending the clinic have lower values for blood vitamin C, even during May and June, when the level of intake is increased (Harris and Olliver, 1943), than the normals set out in Table 1B. As a matter of routine every patient showing a blood vitamin-C level of less than 0·5 mg. per 100 ml., blood was treated with vitamin C to produce saturation as follows: 1st week—4 tablets (50 mg. ascorbic acid each) per day; 2nd week—2 tablets (50 mg. ascorbic acid each) per day; followed by a maintenance dose of 1 tablet per day. The blood was removed in the morning about three hours after a light breakfast of tea and toast. When the patients had been taking vitamin-C tablets they were instructed not to take any tablets for 24 hours before coming for their second blood test, and a fasting sample was removed as before. Of the patients put upon this treatment twenty were re-tested, and all showed an increase of blood vitamin C to 1·0 mg. (or more) per 100 ml. In nine cases levels as high as 1·5 to 1·8 mg. per 100 ml. were reached. In most cases there was clinical improvement, especially where a low blood vitamin-C level was noted in the first place. These clinical symptoms were largely a relief of those symptoms mentioned in the survey—Table 3; but of the greatest effective use was the relief of " tiredness "—a feature which tends to be closely associated with rheumatic toxaemias and causes the most distress in present times. As a result of collaboration and discussion of these data with the physicians in charge of the cases it was evident that saturation with vitamin C causes an improvement in general symptoms, especially noticeable when the condition was associated with a focus of infection.

The effect of a vegetarian diet on the blood level of vitamin C was studied in comparison with that of people on ordinary mixed diets, but exhibiting no symptoms of rheumatism. The results are given in Table 1B, and indicate that a purely vegetarian diet produces levels of blood vitamin C as high as those produced by treating patients with synthetic vitamin C with an intake of 200 mg. daily. This may perhaps lend some support to those who recommend a purely vegetarian diet for rheumatism. It must of course be remembered that the influence of vitamin C in rheumatic diseases is essentially only one aspect of a very much wider problem associated with other nutritional deficiencies or excesses.

A number of the samples of blood reported above were also tested in routine work by the differential sedimentation test (Coke, 1937). The sedimentation value so obtained was correlated with the vitamin-C level in the blood and with the clinical diagnosis at the same time as shown in Table 2.

TABLE 2

Correlation of Blood Vitamin C with Sedimentation Value

Sedimentation Value	1–10%	11–20%	21–30%	31–40%	41–70%
No. of cases ..	20	16	13	10	13
Mean vit. C ..	0·75	0·47	0·53	0·39	0·40
No. of cases above 0·5 mg. per 100 ml.	11	4	8	1	3

Diagnostic Means.—Active infective arthritis (41 cases)—0·49 mg. p. 100 ml. Osteo-arthritis (16 cases)—0·55 mg. p. 100 ml. Non-articular rheumatism (37 cases)—0·58 mg. p. 100 ml.

It was observed that after the patients had been treated with vitamin C the precipitation of blood proteins with metaphosphoric acid was much more efficient, giving crystal-clear solutions quite easily.

A survey was made of 75 patients submitted for vitamin-C tests with a view to ascertaining whether clinical symptoms could be correlated with a partial deficiency (*cf.* Table 3).

TABLE 3

Correlation of Blood Vitamin C with Clinical Symptoms

Symptoms	No. of Cases Showing Blood Vitamin C.		
	Below 0·5 mg. per 100 ml.	0·5–1·0 mg. per 100 ml.	Above 1·0 mg. per 100 ml.
Excessive tiredness ..	31	17	12
Sore gums	7	4	3
Spontaneous bruising	4	5	4
Very easy bruising ..	18	11	7
Anaemia	9	5	1
Endocrine disturbance	6	4	1
Gastric disturbance ..	15	2	7

The figures suggest that there was some positive correlation between low blood vitamin C and the following symptoms: (a) excessive tiredness, (b) tendency to bruising, and (c) gastric disturbances.

Summary

The blood level of vitamin C in 183 cases of chronic rheumatic disease of different types has been determined, the symptoms recorded, and a method of saturation described. An apparent correlation was found between low vitamin-C levels and certain clinical symptoms; also, after saturation with vitamin C an improvement in general health is suggested from the records of clinical observations.

My thanks are due to Dr. Warren Crowe and Dr. Coke for much helpful advice and criticism, to Dr. Coke for the results of the D.S.T. tests in Table 2, to the Medical Staff of the Charterhouse Rheumatism Clinic for sending their patients to the laboratory for these examinations, and to the Council of the Charterhouse Rheumatism Clinic for permission to publish these results and for providing the facilities and means for carrying out the work.

I am indebted to Messrs. Roche Products for their kindness in supplying pure ascorbic acid, and to Messrs. Charles Hearson, Ltd., who designed and made a convenient rack for the gas treatment of a series of samples at the same time.

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FIBROSITIS AND REALITY

BY

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The following personal experience may throw some light on the problems of fibrositis. In March 1935, the writer contracted scarlet fever, the attack was a mild one and recovery appeared to be complete within a few days. Eighteen days after the onset, however, the heels became sore after walking 100 yards on a hard surface with a vivid impression of thickening of the soft tissues, but no swelling was discernible. A few days later spontaneous pains developed in the shoulders, at first inconstant and fleeting but soon becoming more continuous. The pain was felt over the greater tuberosity of the humerus and along the anterior border of each deltoid muscle; no tenderness, swelling or induration of any kind was detected. Bicipital tenosynovitis was diagnosed. If the shoulders were kept at rest little pain was felt for a few minutes; then they would begin to ache and become painfully stiff, and change of position became imperative. Abduction of the humerus was extremely painful for a range of about 30 degrees. The shoulders ached more at night than during the day and were painfully stiff on waking; with use the stiffness would wear off. All movements involving abduction or forward flexion against slight resistance were painful.

Inconstant pains were felt in the lumbar region and the hips and in the knees for a time, but after a few weeks the knees became the seat of a constant disability similar to that in the shoulders, aching more or less continuously, worse at night, the pain being felt mainly on the inner side of the joint from the upper border of the femoral condyle to the lower border of the tibial condyle. There was no tenderness or swelling and no limitation of movement. After walking a few yards weight bearing became painful but was relieved by flexing the joints slightly; unbearable cramps in the thighs occurred if the knees were held in one position continuously. The wrists became painfully stiff so that ordinary movements could not be performed, but again there was no swelling or obvious tenderness.

For five weeks I attempted to carry on, but then went to bed in hospital for a month. The inactivity, however, increased the stiffness without contributing to recovery. Tonsillectomy was performed without any apparent benefit. Diathermy relieved the pain in the knees and shoulders but only for the duration of the treatment, and after it the pain would be worse and the treatment was abandoned after two weeks. A rigor was induced with B.C.C. but without evident improvement. Thirty c.cm. of scarlatinal anti-streptococcal serum were given on admission to hospital; this was followed by general urticaria lasting four days. The temperature was never raised except during the initial attack of scarlet fever and during the artificial rigor. Red and white cell counts were normal, there was no muscular wasting or loss of weight nor any lack of appetite. I had never suffered from rheumatic disease previous to this attack.

I visited a bee farm and allowed myself to be stung on the forearms more than a dozen times without relief. Four weeks after leaving hospital I noticed an improvement in knees and shoulders, and within a fortnight all trace of pain had disappeared; I cannot attribute this recovery to any known cause.

The chief features of this illness were the deep-seated pain without tenderness associated with feebleness and lack of muscular co-ordination and severe cramps, possibly due to irregular muscular contractions of reflex origin. A vivid recollection is the baneful effect of cold in fibrositis, I became extremely sensitive to draughts. This effect was not due to a lowering of body temperature but to increased cold-sensitivity in the skin, and this lasted for several years.

Light Shed by Recent Advances

Lately I have come to regret that I was not at that time possessed of some recently acquired knowledge relating to fibrositic nodules and to referred pain from muscles. I have no doubt that examination by careful palpation would have revealed deep lesions, not in the painful areas but in adjacent muscular tissue. The preliminary work of J. H. Kellgren (1938) suggested that fibrositic lesions had a predilection for certain sites, and that the resultant pain tended to radiate in remarkably regular patterns. This has now been confirmed by a large number of observers, and in many hundreds of cases by the present writer (Kelly, 1941a, b; 1944a, b, c; 1944a, b, c).

The " painful shoulder" comprises a variety of conditions whose detailed description is not my purpose. Suffice to say that in all of them the pain tends to radiate in a constantly recurring pattern.

and that in the majority the offending lesion is situated not in the deltoid but in the muscles attached to the scapula, usually the upper fibres of the trapezius, the supraspinatus, or the infraspinatus. Local anaesthetic injected into this site abolishes the pain, and the relief in a surprising number is permanent (Kelly, 1942b). Almost identical results have been reported by Travell, Rinzler, and Herman (1942).

With fibrositis of the knee the tendency is for the formation of lesions in adjacent soft tissues, usually in relation to the upper border of the internal condyle of the femur or in the calf muscles behind the inner tibial condyle (Kelly, 1944b). Local anaesthetic accurately injected into the right spot will cause an immediate striking amelioration of symptoms; frequently the improvement is lasting. Similar results have been reported by Dally (1940) and Good (1942a, b). Many writers can be quoted who recognized that pain in the knees could proceed from lesions in the soft tissues (Mennell, 1938; Telling, 1935; Kellgren, 1939).

Psychogenic Fibrositis

To different observers fibrositis bears varying degrees of reality. A comprehensive synonym is "non-articular rheumatism"; but the lack of a precise definition of "rheumatism" still leaves the situation far from clarified. Any somatic pain which is not accompanied by well-defined physical signs of disease may therefore come under the general designation of "fibrositis." By some observers, pains which do not conform to certain well-recognized patterns are regarded as psychogenic. Many cases which some physicians diagnose as fibrositis would therefore be considered by others as psychosomatic in origin. Thus to Halliday (1941, 1942) the pain, stiffness, and soreness in the majority of cases are symbolical, signifying the patient's lack of harmony with his surroundings. Ellman, Savage, Wittkower, and Rodger (1942), however, thought that well-defined muscular tenderness indicated an organic basis for the pain, and, where this sign was lacking, they usually were able to elicit a history of psychological upset.

Many others could be quoted. It is in fact almost universal to regard as nervous those pains which cannot be made to harmonize with accepted neurological theory. It has been so for many years; it was noted by the poet Keats more than a hundred years ago that "if the doctors cannot ascertain a disease they call it nervous." But simple assertions will not suffice; a recognizable etiological chain should be established if the psychogenic origin of pain is to be accepted as a scientific fact. Bramwell (1930), who wrote upon psychogenic pain, was taken to task by Walshe (1930), who denied that these so-called pains were pains at all. Pain, says Walshe, cannot even be imagined; what the patient feels in these cases is a mental distress which he incorrectly labels pain.

The Alternatives

Where then lies the truth? When a patient complains of shifting pains for which no cause can be ascertained, we have the option of at least four explanations:

1. The pain symbolizes mental distress, or is resorted to as a refuge from difficulties; therefore it is hallucinatory.
2. The patient mistakenly labels as pain a totally different unpleasant sensation.
3. The patient really has pain, the direct effect of psychic central disturbance upon the body tissues.
4. The patient really has pain, which is not psychogenic but somatic in origin.

I am leaving out the possibility of deliberate simulation, or malingering. Evidence of mental distress is held to enhance the likelihood of a psychogenic origin. It should not be forgotten, however, that long-continued pain of itself will cause mental distress and nervous exhaustion, which then could erroneously be regarded as the cause of the pain. In searching a patient's past for evidence of "psychic trauma," it should be remembered that in the life of almost every normal person there occur mental conflicts which by an enthusiastic psychiatrist might be elevated to a prime cause of psychosomatic disease.

Hallucinations of Pain?

Before accepting as a likely explanation symbolical or hallucinatory pain occasioned by present mental distress or past mental conflicts, we should ask ourselves some questions regarding hallucinations of pain. If they are common in the sane, why do they differ so profoundly in this regard from other hallucinations, which are pathognomonic of insanity or of serious toxic disease? There is a large subjective element in pain which is appreciated only by the sufferer, who very often can produce no evidence of its existence beyond the mere assertion that he feels it. Pain therefore lends itself readily to simulation or exaggeration, but there is no evidence that it thereby becomes more readily the subject of hallucinations or delusions. Solomon (1917), who reviewed the subject at length, concluded that "an idea of a pain can by no manner of means evoke a pain." Walshe (1930) agrees that there is no such thing as subjective pain; he thinks that the patient is suffering from a kind of delusion in interpreting mental distress as pain. In dealing with such intangible matter, however, there would appear to be very little difference between imagining a pain and conjuring up a pain out of a different sort of feeling.

There is no real evidence, in fact, that the vast majority of patients who complain of pain do not actually feel the pain. The onus of disproof is on the observer in every case; he cannot whisk it away by a mere assertion. The fact that the distribution of the pain does not correspond with accepted patterns does not necessarily mean that it has no reality; it is more likely to mean that our knowledge of pain is inadequate. Recent work on deep pain, in fact, suggests that "vague," shifting, poorly

localized pain, accompanied by little anatomical change, may be a feature of many somatic ills. More detailed study of such cases, by questioning and palpation, often reveals constantly recurring patterns of pain-radiation and of tender spots in the deeper tissues. Much that was obscure in fibrositis has been illuminated in the past six years, and one can hopefully expect that further studies, based upon the same scientific foundation, will give greater reality to that which still remains obscure.

Causation

We must assume, therefore, that our patient if he is not malingering really has a pain, " peripherally induced and subjectively appreciated " (Solomon); for want of a better term we label this pain " fibrositis." Very occasionally such an illness begins soon after a severe nervous shock such as a bereavement, or during a period of nervous exhaustion. The question then arises as to the nature of the links in the chain of causation; for all students of rheumatism are agreed that such a succession of events occurs a little too often for mere coincidence. Any type of rheumatic disease, even profound rheumatoid arthritis, may follow severe mental strain.

Considering rheumatic etiology in general, it is probably true that in rheumatic disease of whatever variety the onset can be traced in more than a third of the cases to a particular event, such as infection, injury, or muscle-strain. Thus rheumatic fever frequently follows a sore throat after a silent interval; fibrositis often is brought on by a chill or by an injury; dysentery and many other infectious diseases have recognized rheumatic sequelae; and so on. In my own case, generalized fibrositis followed scarlet fever after a silent period of about a fortnight. There can be no doubt whatever that the fibrositis was etiologically linked to the infection; but the links in the chain are unknown. It is indisputable that the attack of scarlatina must have initiated certain changes in the system which became progressive, and after two weeks manifested themselves as generalized pains. A study of the history of a number of rheumatic cases will reveal a similar chain of events: a general or local infection, an injury or a chill; an injection of T.A.B., or almost any other illness may be the initiating event which sets the pathological process in motion. It is nearly true to say that any form of injury to body cells may be followed by rheumatism. Severe nervous strain comes under this category of injury or insult to the tissues; for exhaustion, great anxiety or grief, or any similar strain, occasions disorder of the cells of the central nervous system even as a chill occasions disorder of the cells of the somatic tissues.

Rheumatism and the Nervous System

In the following case, seen by the writer, organic disorder of the central nervous system was an unmistakable causative factor in joint disease: A man, aged 46, developed Huntington's chorea in its worst form, and died within one year of its onset. During the last six months, while he was bedridden and completely irrational, changes typical of rheumatoid arthritis developed in his knees, ankles, elbows, and wrists. The bone ends became enlarged and tender, with fluid in the joints. Advanced contractural deformities developed, with muscular wasting.

In a similar case reported by de Haas (1941), a patient became deeply cyanotic during anaesthesia and his breathing was arrested. Within a few weeks he developed Parkinsonian tremor, followed by a typical picture of rheumatoid polyarthritis.

If central nervous disease, as in the above cases, is certainly responsible for profound joint changes, it cannot be denied that a related but more transient etiological factor ought sometimes to be responsible for less severe rheumatic symptoms, such as fibrositis. There should be no more mystery, therefore, about fibrositis when it follows nervous exhaustion than when it follows an infection. Rheumatism is etiologically linked to cerebral dysfunction, even as it is linked to infection of the respiratory tract, by a chain of pathological processes whose nature is unknown. The fact that fibrositis sometimes is psychogenic should render it neither more nor less explicable than when it is due to other causes. Once the process has been set in motion, the removal of the original causative factor is by no means certain to reverse it. Thus, in my case, the pains were not felt until some time after I had fully recovered from scarlet fever; fibrositis which follows an injury or exposure to cold will proceed in spite of the healing of the injury or recovery from the chill. Hence the general failure to cure rheumatism by removal of foci of infection. The infection which initiates the rheumatic process is not of necessity instrumental in its continuance, for an additional unknown factor has begun to act.

Similarly, when rheumatism follows nervous exhaustion the disorder of the cells has initiated a process which then comes under the control of unknown factors. Recovery of the nerve cells (whether in the course of nature or with the aid of psychotherapy) could not always be expected to result in the cure of the rheumatic process, even as recovery from a sore throat has no relation to the cessation of an attack of rheumatic fever.

If this thesis be true, the fibrositis which follows nervous exhaustion is just as real as that which follows any other cause, or the cause of which is unknown. The uncovering of a psychic cause will render the disease neither more nor less explicable, and the disappearance of the nervous disorder should not be expected to bring about certain cure. The psychogenic factor, in order to be significant, should be as unmistakable as other etiological factors in other cases; strains and conflicts which require analysis to reveal them are too obscure and intangible to have any certain validity. In this regard there is a close analogy to physical strain, where the line of demarcation between normal use and overstrain of muscles is quite indefinite. In many a case, when an attack of fibrositis suddenly begins during an ordinary movement it is impossible to say to what extent the movement has been the causative factor.

In like manner, chronic rheumatic disease is often seen in parts which have been subjected to long-continued over-use, as the shoulders of labouring men; and, in a still greater number of cases seen in middle life, it is quite difficult to say to what extent the prolonged physiological use incidental to ordinary activity has been a contributing factor. Similarly with mental stress—the business of life normally carries with it a succession of conflicts, strains, and processes of adjustment, and in the majority of cases it is impossible to say whether experiences normal to the average individual have or have not been injurious to the patient.

The Need for Facts

The attack of fibrositis experienced by the writer possessed many of the attributes regarded as typical of psychosomatic disease; in fact, a colleague frankly regarded the affliction as a neurosis. Prior to its onset, however, I had been completely free from anxiety or mental stress of any kind; later, when I was experiencing some anxiety about the future my inexplicable recovery took place.

Whenever I read of psychological explanations of fibrositis, my own experiences come to mind. My conviction, based on personal experience, is that the " vague " pains of which so many patients complain have more reality than is usually attributed to them. A debt of gratitude is due, no doubt, to the teachers who have traced the psychosomatic relationship; but the symptoms can be explained upon the basis of known somatic reactions rather than of mystic symbolism or emotional delusions.

Of all kinds of disease, the rheumatic group is the one on which modern scientific progress has made least impression. Little indeed is known, and yet we are possessed of a few facts. An outstanding fact is that these diseases are painful. If we move away from this basis of reality we may wander far into the wilderness. If our theories relating to pain do not accord with observed facts, 'twere wiser to modify the theories rather than to explain away the facts.

Summary

1. A personal experience is described in which, following a mild attack of scarlatina, the writer suffered for three months from generalized pains and muscular stiffness, entirely unaccompanied by physical signs.

2. Prevalent views upon psychogenic fibrositis are considered in the light of the writer's experiences. The idea of psychogenic pain should not be divorced from reality, for all pains are peripherally induced and subjectively appreciated.

3. When rheumatic pain follows nervous exhaustion the etiological relationship differs little, if at all, from the case when it follows somatic disease or injury.

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